THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM PCP

Using the Recovery Kernel Framework

PCP · Phencyclidine · Angel Dust · Wet · Sherm · Embalming Fluid · Rocket Fuel · Supergrass · Hog · Boat

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

PCP INTOXICATION IS A MEDICAL EMERGENCY. AGITATION + PAIN INSENSITIVITY + PSYCHOSIS IS THE SPECIFIC CLINICAL PICTURE. CALL 911.

PCP PSYCHOSIS CAN PERSIST FOR WEEKS AFTER THE LAST DOSE. IF SYMPTOMS DO NOT CLEAR IN 48 HOURS: PSYCHIATRIC EVALUATION.

The compendium names CYP2B6 as the Dissociative Logjam gene. PCP is its most clinically dangerous substrate.

A Note Before You Begin

PCP (phencyclidine) is the most pharmacologically complex substance in this guide series. Every other dissociative covered here — ketamine, DXM, nitrous oxide — operates primarily through a single dominant mechanism. PCP operates through four simultaneously, and the specific combination of those four produces a clinical picture that has no equivalent in any other substance of misuse.

NMDA receptor antagonism so potent that it rivals surgical anesthesia at high doses. Dopamine reuptake inhibition producing stimulant arousal and agitation. Sigma-1 receptor agonism producing the specific perceptual alterations unique to this compound class. Muscarinic acetylcholine antagonism producing the anticholinergic features — dry mouth, elevated heart rate, urinary retention, hyperthermia — that compound the other three.

The result: a person who is simultaneously profoundly disconnected from reality (NMDA), highly aroused and potentially agitated (dopaminergic), experiencing perceptual phenomena unlike any other class (sigma-1), and physiologically stressed by anticholinergic load. This combination — dissociation and stimulation together, pain insensitivity and high arousal together — is what produces the

specific and genuinely dangerous behavioral picture of PCP intoxication that no framework built for any other substance adequately addresses.

The Recovery Kernel Compendium names CYP2B6 as the 'Dissociative Logjam Risk' gene. PCP is the most clinically consequential substrate for this gene in common substance misuse. A CYP2B6 poor metabolizer encountering PCP is entering a pharmacokinetic situation where the most potent commonly used NMDA antagonist known cannot be cleared — accumulating at the receptor, extending and deepening both the dissociation and the psychosis beyond anything the person's dose would produce in a normal metabolizer.

This guide addresses PCP recovery specifically. The ketamine guide in this series addresses adjacent territory — the NMDA antagonist recovery framework, the glutamate rebound, the dissociative natural porting architecture. Read it alongside this guide for the NMDA dimension. What this guide adds is what makes PCP specifically different from ketamine — the dopaminergic stimulant component, the anticholinergic load, the extended duration, the dramatically higher psychosis liability, and the specific clinical and community context that has made PCP one of the most misunderstood substances in addiction medicine.

Section 0 — Immediate Safety: The PCP Intoxication Emergency

⚠ THE SPECIFIC CLINICAL PICTURE OF PCP INTOXICATION — STIMULANT AGITATION + NMDA PAIN INSENSITIVITY + DISSOCIATIVE PSYCHOSIS — IS UNIQUELY DANGEROUS. STANDARD APPROACHES TO MANAGING INTOXICATED INDIVIDUALS DO NOT APPLY. READ THIS SECTION BEFORE ANYTHING ELSE.

0.1 — Recognizing PCP Intoxication

THE CENTURION — The Four Signs That Together Point to PCP — Not Any Other Substance

PCP intoxication produces a specific combination that distinguishes it from stimulant intoxication, opioid overdose, and pure dissociative states:

Sign	What it looks like	The mechanism
Nystagmus — rapid, involuntary eye movement	The eyes move rapidly from side to side, up and down, or in rotary patterns. The person cannot hold their gaze on a fixed point. This is the single most reliable clinical sign of PCP intoxication and distinguishes it from most other substances.	NMDA antagonism in the vestibular and oculomotor systems. Nystagmus is so consistently present that its absence makes PCP unlikely. Emergency clinicians use it as a rapid identification tool.
Analgesia — complete or near-complete pain insensitivity	The person does not respond to pain or responds minimally. They may sustain significant injuries — burns, lacerations, fractures — and appear not to feel them. This is NOT voluntary. It is NMDA-mediated analgesia identical to what makes PCP useful in veterinary surgery.	NMDA receptors are central to pain processing in the dorsal horn of the spinal cord. PCP's potent NMDA blockade provides complete analgesia. The clinical danger: the person cannot use pain as a warning signal that they are injuring themselves.
Agitation and stimulant arousal	Unlike ketamine or DXM intoxication (which typically produces sedation), PCP intoxication frequently produces extreme agitation, hyperactivity, apparent strength beyond normal capacity, combativeness, and dysphoric arousal. The person is not calm — they are highly activated.	PCP's dopamine reuptake inhibition — the mechanism absent in ketamine — produces stimulant arousal alongside the NMDA dissociation. The combination of high arousal and complete disconnection from reality is what produces the dangerous behavioral profile that PCP is uniquely associated

		with.
Psychosis and profound disorientation	The person does not know where they are, who they are with, or what is happening. They may be responding to internal stimuli (hallucinations), making grandiose statements, or expressing paranoid terror. Thought is profoundly disorganized.	NMDA antagonism in the prefrontal cortex and limbic system produces both positive psychotic symptoms (hallucinations, delusions) and the profound disorientation that distinguishes PCP from stimulant psychosis. PCP-induced psychosis is the most valid pharmacological model of schizophrenia currently known — it produces both positive AND negative symptoms that amphetamine psychosis does not.

0.2 — The Emergency Response Protocol

⚠ DO NOT ATTEMPT TO PHYSICALLY RESTRAIN A PCP-INTOXICATED PERSON AGGRESSIVELY. THE CATECHOLAMINE SURGE FROM FORCIBLE RESTRAINT PRODUCES HYPERTHERMIA AND CARDIAC RISK ON THE ALREADY STRESSED CARDIOVASCULAR SYSTEM. THIS HAS RESULTED IN DEATHS.

Priority	Action	Why
1 — Call 911	Call first. Do not wait to see if the person 'comes down.' PCP intoxication is a medical emergency requiring EMS assessment. Tell the dispatcher specifically: 'PCP intoxication — phencyclidine — with agitation and apparent psychosis.' This changes the emergency response sent.	EMS providers trained for PCP will use chemical sedation (benzodiazepines) rather than physical restraint as their primary approach. This is the safest and most effective management and requires the right clinical team.
2 — Low stimulation	Reduce all sensory input. No bright lights, no loud voices, no sudden movements, no touching. A calm, quiet, dark environment is the most effective environmental intervention available. Speak slowly, in a low voice, from a distance.	Sensory stimulation in PCP intoxication amplifies the agitation and the psychotic features. The sigma-1 and dopaminergic components of PCP make the person hyperresponsive to sensory input. The 'talk-down' approach that works for psychedelics does not work for PCP — but sensory reduction does help.
3 — Safe space	Remove the person from traffic, water, heights, and any environment where the analgesia	The analgesia of PCP means the normal protective reflexes are absent. A person who is PCP-

	(pain insensitivity) allows them to sustain injury without awareness. A padded, low-stimulation room is the ideal environment pending EMS arrival.	intoxicated can walk into traffic, fall from heights, or sustain burns without responding. Environmental safety is a primary responsibility.
4 — Benzodiazepines	In the hospital or EMS setting: benzodiazepines (diazepam 10mg IV or IM, lorazepam 2mg IM) are the first-line pharmacological management for PCP agitation. They address both the dopaminergic agitation and the NMDA-mediated features.	Benzodiazepines are more effective and safer than antipsychotics as first-line for PCP agitation. Antipsychotics lower the seizure threshold (a PCP risk), can worsen anticholinergic effects, and do not address the NMDA component as effectively as benzodiazepines.
5 — Temperature monitoring	PCP produces hyperthermia through the dopaminergic stimulant mechanism and the anticholinergic sweat suppression. Core body temperature monitoring. Aggressive cooling if temperature exceeds 39°C (102°F).	Hyperthermia is a primary mortality mechanism in PCP toxicity. The combination of stimulant metabolic demand (dopaminergic), impaired heat dissipation (anticholinergic anhidrosis), and the catecholamine surge of agitation produces rapid temperature elevation.

0.3 — The Prolonged Psychosis Warning

⚠ PCP PSYCHOSIS CAN PERSIST FOR DAYS TO WEEKS AFTER THE LAST DOSE — FAR LONGER THAN ANY OTHER NON-PSYCHEDELIC SUBSTANCE IN THIS GUIDE SERIES. THIS IS NOT 'STILL HIGH.' IT IS A SPECIFIC NEUROBIOLOGICAL STATE REQUIRING CLINICAL MANAGEMENT.

PCP's slow dissociation from the NMDA receptor — combined with its lipophilicity (stored in body fat and slowly released) — produces psychotic symptoms that persist long after the drug has cleared plasma. For CYP2B6 poor metabolizers, the accumulation problem extends this further. The clinical picture can meet DSM-5 criteria for a brief psychotic disorder, schizophreniform disorder, or even schizophrenia during the active phase — none of which it is.

- If psychosis does not substantially resolve within 48 hours of last PCP use: psychiatric evaluation is required. Prolonged PCP psychosis is a medical condition, not a recovery state.
- Antipsychotic medication is the indicated treatment for prolonged PCP psychosis — risperidone, olanzapine, or quetiapine. Do not wait to see if it resolves without treatment; each day of untreated psychosis carries its own risks.
- Inform the treating psychiatrist specifically: 'This is PCP-induced psychosis, not primary psychosis.' The treatment and prognosis are different — PCP psychosis resolves with sustained abstinence in most cases; primary schizophrenia does not. This distinction affects both immediate medication decisions and long-term management.

- Sleep is the most powerful non-pharmacological intervention for PCP psychosis. Inducing sleep through benzodiazepines or quetiapine often produces dramatic improvement upon waking.

0.4 — What Is Actually in 'Wet' / 'Sherm' — The Adulteration Problem

THE CENTURION — The Urban Legend vs. The Pharmacology — Embalming Fluid Is the Delivery Vehicle, Not the Drug

'Wet' or 'sherm' — cannabis or tobacco cigarettes dipped in a liquid and dried — is the most common contemporary PCP use method. The name 'embalming fluid' has generated a persistent urban legend that the cigarette is dipped in formaldehyde. This is incorrect as the active pharmacology. The liquid is either pure PCP in solution or PCP dissolved in an organic solvent, sometimes including real embalming fluid as the solvent base.

The clinical implications:

- Formaldehyde (if present as a solvent component) adds pulmonary and systemic toxicity on top of PCP's pharmacological effects — making the combination more dangerous than PCP alone
- The PCP concentration in wet cigarettes is entirely variable — there is no standardized dose. What feels like 'one hit' can contain dramatically different amounts of PCP depending on the preparation
- Street verification: there is no reliable field test for PCP in wet cigarettes. The DanceSafe/reagent testing approach (Marquis reagent turns pink-orange with PCP) can identify PCP's presence but not concentration
- What is sold as wet/sherm frequently contains other substances — ketamine, PCP analogs (PCE, TCP, PCC), or no PCP at all

Section 1 — What PCP Did to Your Brain

THE CENTURION — The Only Widely Used Substance That Is Simultaneously a Powerful Dissociative AND a Powerful Stimulant

PCP was developed in the 1950s as a general anesthetic (marketed as Sernyl) — an agent that produced surgical anesthesia through dissociation rather than unconsciousness. Its use was discontinued in humans because too many patients experienced severe emergence reactions: profound agitation, hallucinations, and delirium upon waking. These are identical to the effects that make it a drug of misuse.

Its veterinary use (as Sernylan) continued because animals do not have the same post-dissociative emergence reaction. It remains in use today as a large-animal veterinary anesthetic under the name 'Rompun' — wait, that is xylazine. PCP is rarely used veterinarily now. Its primary contemporary existence is as an illicit substance.

What makes PCP pharmacologically unique across the entire landscape of substances of misuse is the specific combination of receptor targets it activates simultaneously:

1.1 — The Four-Receptor Architecture

Receptor system	What PCP does	Recovery implication
NMDA glutamate receptors (primary — defining)	PCP is one of the most potent NMDA receptor antagonists in common use — significantly more potent than ketamine or DXM at the same plasma concentrations. It enters the ion channel when it opens and blocks it from inside (open-channel block), but it dissociates from the receptor far more slowly than ketamine. This 'trapping' within the open channel means that even as plasma PCP levels fall, the receptor remains blocked. The result: longer duration of NMDA blockade than plasma levels would predict.	NMDA rebound on cessation is more prolonged and more severe than ketamine withdrawal. The glutamate surge when NMDA receptors recover from PCP blockade contributes to the anxiety, cognitive disorganization, and excitotoxic stress of PCP cessation. NAC and magnesium are the primary management tools — same as ketamine and DXM recovery but requiring more sustained application.
Dopamine transporter (DAT) — stimulant component	PCP inhibits dopamine reuptake through the DAT — the same mechanism as cocaine. This is what makes PCP fundamentally	The dopamine reconstruction protocol from the stimulant recovery guides (cocaine, meth) applies as a secondary track in

	different from ketamine and DXM, neither of which significantly affect the dopamine transporter. The dopaminergic component produces stimulant arousal, euphoria, agitation, and the high addiction liability that pure NMDA antagonists like ketamine do not produce as readily.	PCP recovery. Tyrosine, the Brain Stack (UMP + DHA + CDP-Choline for D2 receptor upregulation), and NAC address the dopaminergic dimension. The Warrior COMT profile is relevant here as it is for all dopaminergic substances.
Sigma-1 receptors	Like DXM, PCP is a sigma-1 receptor agonist. Sigma-1 receptor activation produces the specific perceptual and mood alterations that distinguish PCP from pure dopaminergic stimulants or pure NMDA antagonists — a dissociative quality layered with the perceptual disturbances that sigma-1 agonism produces.	The sigma-1 component of PCP recovery is addressed similarly to DXM recovery — mood and perceptual normalization over weeks of abstinence. The recovery of sigma-1 receptor sensitivity is supported by the general neuroprotective and antioxidant stack.
Muscarinic acetylcholine receptors (anticholinergic)	PCP blocks muscarinic acetylcholine receptors, producing anticholinergic effects: dry mouth, tachycardia, urinary retention, impaired sweating (dangerous for hyperthermia), dilated pupils, confusion. This is the fourth mechanism that ketamine and DXM do not share to the same degree.	The anticholinergic component of PCP must be considered in acute management (impaired sweating compounding hyperthermia; urinary retention requiring catheterization if severe) and in recovery (choline supplementation to support the acetylcholine system that has been antagonized). Alpha-GPC or CDP-Choline are the primary acetylcholine recovery supplements.

1.2 — PCP vs. Ketamine: The Clinical Differences That Matter for Recovery

THE CENTURION — Same NMDA Mechanism. Completely Different Clinical Picture.

Variable	PCP	Ketamine (for comparison)
Primary additional mechanisms beyond NMDA	DAT inhibition (dopaminergic stimulant) + sigma-1 agonism + muscarinic antagonism (anticholinergic)	Some sigma-1 activity; no significant DAT inhibition; no significant muscarinic antagonism
Duration at typical recreational doses	4–8 hours primary effects; residual psychotic features can	1–2 hours (intranasal/IM); effects substantially resolved by

	persist days to weeks	3–4 hours in most cases
NMDA receptor dissociation speed	Very slow — PCP is 'trapped' inside the open NMDA channel, taking much longer to dissociate than ketamine. This produces longer-lasting receptor blockade at equivalent initial binding.	Faster — ketamine dissociates from the receptor more readily, producing the shorter and more titratable clinical effects that make it manageable in anesthetic settings.
Behavioral profile during intoxication	Agitation, combativeness, apparent elevated strength, nystagmus, analgesia, psychosis. The stimulant + dissociative combination is what produces the unique behavioral picture.	Sedation, dissociation, analgesia, emergence phenomenon (brief confusion). Agitation is possible but less common and less severe than PCP. The 'K-hole' is profound sedation, not stimulant agitation.
Addiction liability	Higher than ketamine — the dopaminergic stimulant component adds the reward circuit activation that drives conventional addiction. PCP produces stronger dopamine-mediated wanting than ketamine.	Lower conventional addiction liability — primarily through NMDA/sigma mechanisms without significant dopaminergic reward. Psychological dependence on the dissociative state is more prominent than dopaminergic craving.
Psychosis risk	Very high — PCP-induced psychosis is the best pharmacological model of schizophrenia known to researchers because it produces both positive AND negative symptoms. Prolonged psychosis after cessation is a specific and common complication.	Moderate — ketamine can produce psychotic features at high doses and with chronic use, but prolonged post-use psychosis lasting weeks is much less common than with PCP.
Route of use	Primarily smoked (wet/sherm), insufflated, or oral. The smoked route (wet cigarettes) is the most common contemporary method and adds pulmonary exposure and very fast onset to the risk profile.	Primarily insufflated (powder) or IM injection (pharma grade). The intranasal and IM routes provide more predictable absorption than the wet cigarette.

1.3 — The PCP Psychosis Model: Why This Matters for Recovery

THE CENTURION — The Brain on PCP Is the Closest Available Model of Schizophrenia. Recovery Requires Understanding This.

Since the 1960s, PCP has been recognized as the most pharmacologically valid model of schizophrenia available to researchers. Amphetamine psychosis produces positive symptoms (hallucinations, paranoid delusions) but not the negative

symptoms (emotional blunting, poverty of thought, social withdrawal) that characterize schizophrenia's full clinical picture. PCP produces both.

This is not merely interesting neuroscience. It has direct recovery implications:

- PCP psychosis requires antipsychotic treatment, not just abstinence. Waiting for it to resolve without treatment is both uncomfortable and carries its own risks from the agitated and self-endangering behavior it produces.
- PCP psychosis can be misdiagnosed as schizophrenia. A first-episode psychosis presentation in a young person whose history includes PCP use should always consider PCP as a causative factor before diagnosing a primary psychotic disorder. Urine drug screens may be negative by the time of psychiatric evaluation (PCP is detectable for 7–30+ days depending on use pattern, but clinical presentation may occur early in this window).
- The negative symptoms of PCP psychosis — emotional blunting, withdrawal, cognitive poverty, decreased motivation — look and feel like depression to the person experiencing them and to clinicians not considering PCP. This can result in incorrect treatment (antidepressants for PCP psychosis negative symptoms) when antipsychotics and abstinence are the indicated interventions.
- For heavy, long-term PCP users: a persistent deficit state may be present even after psychosis resolves — the cumulative NMDA antagonism producing lasting changes in prefrontal cortex function. This is the 'PCP-induced cognitive impairment' that may require extended support and is addressed in Section 4.

1.4 — What PCP Was Providing: The Signal

The compendium's C-37 substance masking analysis: every substance preference is a signal. PCP's four-receptor profile means it was providing multiple things simultaneously:

What PCP provided	The underlying deficit	Recovery address
Dissociative escape from ordinary consciousness	The same deficit as ketamine — the DMN suppression and ego dissolution that the compendium identifies as accessible through meditation. Depression, trauma, chronic anxiety, the unbearable quality of ordinary waking experience.	Section 6: contemplative practice, NSDR, holotropic breathwork as the natural DMN suppression architecture. The ketamine guide's natural porting framework applies here directly.
Stimulant intensity and arousal	The same Warrior COMT deficit as cocaine and meth — the Reward Deficiency that makes ordinary dopamine stimulation inadequate. PCP's dopaminergic	The stimulant recovery natural architecture: vigorous competition, mastery, genuine high-stakes achievement. Same framework as cocaine and

	component provided the intensity the Warrior baseline requires.	methamphetamine recovery for this component.
Pain relief — physical and emotional	PCP's NMDA-mediated analgesia provides genuine and complete pain relief. People with chronic pain, trauma-related hypervigilance (where the pain system is perpetually activated), or the physical pain of difficult life circumstances found in PCP a complete pain relief that other substances approximated but did not match.	Pain management track — same as the kratom and fentanyl guides. Non-opioid pain management, physical therapy, the endorphin and exercise architecture. The NMDA pain relief can be partially approximated through the natural NMDA modulation of magnesium and low-dose approaches.
Social and economic context — availability and affordability	In the communities most affected by PCP — historically urban, lower-income, predominantly Black — PCP has been available, affordable, and embedded in social networks in ways that more expensive or less available substances were not.	Same structural recovery acknowledgment as the crack cocaine guide. The economic and community dimensions of PCP use require structural response alongside neurochemical recovery.

1.5 — Duration, Metabolism, and the Lipophilicity Problem

PCP is highly lipophilic — it dissolves readily into fatty tissue and distributes extensively throughout the body. This produces several clinically important pharmacokinetic features:

- Long half-life: PCP has a plasma half-life of 7–46 hours (highly variable depending on CYP2B6/CYP3A4/CYP2C19 metabolizer status and urine pH). The long half-life compounds the extended duration of effects.
- Urine pH effect: PCP is excreted in urine in a pH-dependent manner. Acidic urine dramatically increases PCP excretion. This was historically used in emergency management (ammonium chloride to acidify urine and accelerate PCP clearance) but is now rarely practiced due to complications.
- Fat tissue storage: PCP accumulates in adipose tissue during heavy use. During periods of fat mobilization (exercise, caloric restriction, illness), stored PCP is released into the bloodstream. This can produce unexpected re-emergence of PCP effects — including psychotic symptoms — days or weeks after apparent cessation. The compendium names this phenomenon in the ketamine phantom smell section: 'the body is literally processing residue that was stored in fat tissue during active use.'
- Urinary detection: PCP is detectable in urine for 7 days in occasional users and up to 30+ days in heavy users, with some cases of positive tests at 4–6 weeks after last use due to fat tissue release.

Section 2 — Your Genomic Blueprint

PCP has a pharmacogenomic profile that combines the dissociative logjam risk (CYP2B6) named in the compendium with the dopaminergic stimulant genetics (COMT, DRD2, DAT1) and the psychosis risk profile (COMT, DRD2) that the stimulant guides identify. It is the most genetically complex recovery profile of any single substance in this series.

Gene	What it controls	Key variant	PCP recovery relevance
CYP2B6	One of the primary PCP metabolism enzymes — contributes to PCP clearance through oxidative N-demethylation and hydroxylation pathways	*6/*6 poor metabolizer (rs3745274); *1/*6 intermediate; *1/*1 normal	THE gene the compendium flags as the 'Dissociative Logjam Risk' for PCP and related dissociatives. CYP2B6 *6/*6 poor metabolizers cannot adequately clear PCP — it accumulates at the NMDA receptor far longer than in normal metabolizers. The prolonged PCP psychosis that distinguishes PCP from ketamine is most severe in CYP2B6 poor metabolizers. If PCP effects were consistently more intense, more prolonged, or more psychotically severe than peers at equivalent doses: CYP2B6 poor metabolizer profile is the most likely explanation. This information is critical for any future medication encounter involving CYP2B6-metabolized drugs (efavirenz, bupropion, cyclophosphamide, several psychiatric medications).
CYP3A4	Primary hepatic PCP metabolism pathway — contributes alongside CYP2B6 to PCP clearance	Inducers vs. inhibitors	CYP3A4 inhibitors (fluconazole, many antibiotics, grapefruit, some HIV ARTs) slow PCP clearance by blocking the primary metabolic pathway. Combined with poor CYP2B6 status: the patient whose two primary PCP clearance pathways are both impaired faces extreme accumulation. CYP3A4 inducers (rifampin, carbamazepine) accelerate clearance — historical use of urine acidification was partly trying to compensate for what CYP induction can help achieve through the hepatic route.
COMT	Dopamine	Val158Met	COMT is relevant to PCP through two

	clearance speed — the Warrior/Sage constitution, with specific relevance to PCP psychosis risk	(rs4680) — Val/Val (Warrior) for dopamine primary; Met/Met (Sage) for psychosis risk	distinct mechanisms. (1) Dopaminergic vulnerability (Val/Val Warrior): the Warrior's intensity needs drove the PCP use for its dopaminergic stimulant component alongside the dissociation. Recovery architecture must address the Warrior constitution. (2) Psychosis risk (Met/Met Sage): elevated baseline catecholamine tone combined with PCP's dopamine reuptake inhibition and NMDA antagonism creates the highest psychosis risk profile. Sage constitution + PCP is the combination that most frequently produces prolonged, severe psychosis. The compendium's Sage Worrier architecture is a risk factor, not a failure.
DRD2 / ANKK1	D2 receptor density — Reward Deficiency Syndrome; also relevant to psychosis risk through dopamine D2 pathway	Taq1A — A1 allele (reduced D2 density)	DRD2 A1 allele produces reduced D2 receptor density — the same Reward Deficiency Syndrome as in cocaine, meth, and crack cocaine. PCP's dopaminergic stimulant component was filling the same D2 deficit. Brain Stack (UMP + DHA + CDP-Choline) for receptor density upregulation is the structural recovery target. D2 receptor downregulation from chronic PCP's dopaminergic component compounds the baseline A1 allele deficit.
MTHFR	Methylation — dopamine, serotonin, and neurotransmitter synthesis foundation	C677T, A1298C	5-MTHF addresses the baseline synthesis deficit that PCP was compensating through its pharmacological flooding. Relevant for both the dopaminergic and the NMDA-adjacent serotonin dimensions of PCP recovery. Often the most impactful early supplement for mood and cognitive recovery.
BDNF	Neuroplasticity and prefrontal repair following NMDA antagonist-induced damage	Val66Met (rs6265) — Met allele reduces BDNF secretion	PCP's prolonged NMDA antagonism produces more significant prefrontal BDNF suppression than ketamine due to the longer receptor occupation. Met allele carriers have reduced BDNF secretion, potentially facing slower cognitive recovery from PCP's prefrontal cortex effects. Semax (BDNF-stimulating peptide) and vigorous daily exercise are the primary BDNF interventions — both

			critical for Met carriers.

Phenotyping Without Testing

- Were PCP effects consistently more intense, more prolonged, or more psychotically severe than others at the same dose? → CYP2B6 *6/*6 poor metabolizer very likely. The dissociative logjam — PCP accumulating without adequate clearance.
- Did psychosis or confusion persist for days after the last use? → CYP2B6 poor metabolizer status OR fat tissue storage releasing PCP during fat mobilization — or both.
- Was the primary pull of PCP the stimulant intensity rather than the dissociation? → Warrior COMT constitution + DRD2 A1 Reward Deficiency Syndrome. Stimulant recovery architecture is the primary natural porting target.
- Was the primary pull the escape, the disconnection, the pain relief? → NMDA dissociative architecture is the primary target. Contemplative and trauma-processing framework.
- Did PCP make you feel 'calm' or 'right' in a way that other stimulants didn't? → Possible Sage COMT profile finding in PCP's NMDA quieting of the overactive prefrontal system the relief the Sage's overloaded catecholamine system could not otherwise access. Also the highest psychosis risk profile.

Section 3 — Medical Management: What PCP Recovery Requires Clinically

THE CENTURION — PCP Recovery Has Medical Dimensions That Other Dissociatives Do Not — Because of the Prolonged Psychosis Risk

Unlike ketamine recovery or DXM recovery — where medical intervention in early cessation is rarely required beyond the standard supplement and behavioral protocol — PCP recovery has a specific medical dimension: the management of prolonged PCP psychosis. This requires clinical partnership that most other guides in this series do not.

3.1 — Managing Prolonged PCP Psychosis

Duration and clinical presentation	Indicated management	Notes
Acute psychosis (hours to 48 hours)	Benzodiazepines first-line: diazepam 10mg oral/IM or lorazepam 2mg IM. Low-stimulation environment. Forced sleep if possible — sleep is the most effective single intervention.	Antipsychotics are second-line for acute PCP, not first. Haloperidol lowers seizure threshold. Benzodiazepines address both the dopaminergic agitation and the NMDA component more effectively.
Prolonged psychosis (48 hours to weeks)	Atypical antipsychotic: quetiapine (25–200mg daily, titrate) or olanzapine (5–20mg daily). These have lower extrapyramidal risk than typicals. Quetiapine has the additional benefit of supporting sleep.	Psychosis prognosis: most PCP psychosis resolves substantially within 2–6 weeks of abstinence with appropriate antipsychotic support and sleep. CYP2B6 poor metabolizers may take longer due to slower PCP clearance. Document the timeline — if psychosis persists beyond 6 weeks of full abstinence, independent psychotic disorder evaluation is warranted.
Persistent negative symptoms (emotional blunting, motivation deficit, cognitive poverty)	These may persist after positive psychotic symptoms resolve. They resemble depression but are dopaminergic-negative in origin (D2 receptor downregulation and prefrontal BDNF suppression) rather than serotonergic. Exercise, BDNF building (Semax, lion's mane),	Do not prescribe antidepressants as the primary treatment for PCP-related negative symptoms without considering the dopaminergic mechanism. They may help if there is concurrent serotonergic depression, but the negative symptoms themselves require

	and the dopamine reconstruction stack are the primary interventions alongside antipsychotics where ongoing.	the dopamine and BDNF recovery architecture.

3.2 — Having the Physician Conversation

PCP use disorder is poorly known among general practitioners and even many addiction medicine specialists. The clinical framing that produces appropriate response:

'I have a history of phencyclidine (PCP) use disorder. PCP is a potent NMDA antagonist with additional dopamine reuptake inhibition, sigma-1 agonism, and muscarinic antagonism. I may have CYP2B6 poor metabolizer status based on my response to the drug. I am currently experiencing [symptom]. I am requesting CYP2B6 and CYP3A4 metabolizer testing. I am also requesting evaluation for PCP-induced cognitive impairment and [psychosis management / sleep support / other] through appropriate pharmacological management.'

The physician who does not know about the CYP2B6 dissociative logjam needs to know about it — not only for current management but because CYP2B6 metabolizer status affects the safety of several medications they may prescribe (efavirenz for HIV, bupropion for depression or smoking cessation, cyclophosphamide if oncological treatment is ever needed).

3.3 — No FDA-Approved MAT for PCP

Like cocaine, methamphetamine, and DXM, PCP has no FDA-approved medication-assisted treatment. The pharmacological support for PCP recovery is:

- Antipsychotics for prolonged psychosis (as above — not MAT, but specific medical management)
- Benzodiazepines for acute anxiety and sleep disruption in early cessation (short-term only, addiction risk in this population)
- Non-stimulant ADHD treatment (atomoxetine, guanfacine) if ADHD co-occurs — the DAT-mediated stimulant component of PCP may have been self-medicating
- Depression treatment: the negative symptom presentation distinguishes from serotonergic depression — identify which dimension is dominant before selecting treatment. 5-MTHF first for MTHFR variants.

Section 4 — Biological Reconstruction

PCP biological reconstruction combines three parallel tracks: (1) NMDA glutamate system normalization (ketamine/DXM guide framework), (2) dopaminergic stimulant system reconstruction (cocaine/meth guide framework), and (3) acetylcholine system support for the anticholinergic component. The methylation foundation underlies all three.

4.1 — The Methylation Foundation

Supplement	Dose	Timing	PCP-specific notes
5-MTHF (L-methylfolate)	400–1000mcg	Morning	PCP's multi-receptor disruption impairs neurotransmitter synthesis across dopamine, serotonin, and acetylcholine systems. 5-MTHF provides the methylation foundation for all three through the same BH4 and SAMe pathways that support synthesis throughout. Often the most impactful early supplement for mood and cognitive recovery.
Methylcobalamin (B12)	1000–2000mcg sublingual	Morning	Active B12 for methylation cycle support. Particularly relevant if the PCP use pattern involved poor nutrition, as heavy PCP users frequently experience nutritional neglect during binge periods.
P5P (active B6)	25–50mg	Morning	Cofactor for dopamine, serotonin, and GABA synthesis — covering all three systems disrupted by PCP's complex pharmacology.
Magnesium glycinate	400–600mg elemental	Morning and evening	The endogenous NMDA channel modulator. As in ketamine and DXM recovery: magnesium provides gentle, natural NMDA modulation that eases the glutamate rebound of NMDA antagonist cessation. Begin Day 1. The most immediately effective supplement for the anxiety and cognitive hyperexcitability of PCP

			cessation.

4.2 — NMDA Glutamate System Normalization

- NAC (600–1800mg daily, divided, with food): glutamate homeostasis in the nucleus accumbens. Multiple mechanisms relevant to PCP recovery — craving reduction, antioxidant, and direct glutamate modulation. Begin Day 1. The most multi-functional supplement for PCP recovery.
- L-Theanine (200–400mg, morning and evening): NMDA receptor glycine site modulation; anxiolytic; alpha-wave promotion. Addresses the cognitive hyperactivity of NMDA rebound.
- Glycine (3–5g, evening): NMDA co-agonist supporting receptor normalization during recovery. The ketamine guide's glycine rationale applies: supporting some NMDA activity helps the receptor system normalize after chronic antagonism.
- Omega-3 DHA (2–4g daily): neuronal membrane support for NMDA receptor environment; anti-neuroinflammatory. The most persistent source of neuroinflammation in PCP recovery is the prolonged NMDA antagonism and the oxidative stress of heavy PCP use.

4.3 — Dopaminergic Stimulant System Reconstruction

- L-Tyrosine (500–2000mg, morning, away from protein): dopamine precursor for the DAT-inhibition component of PCP. The same primary reconstruction supplement as cocaine and meth recovery.
- Brain Stack — Uridine (250mg) + DHA (1g) + CDP-Choline (250mg): D1/D2 receptor density upregulation. The most important structural recovery target for the DRD2 A1 Reward Deficiency Syndrome that PCP was feeding through its dopaminergic component. Takes weeks to months; begin Day 1.
- Rhodiola rosea (300mg, morning): dopamine and serotonin modulation; adaptogenic anti-fatigue. Particularly useful in Weeks 2–8 when the dopaminergic deficit produces the 'grey' quality of early stimulant recovery.

4.4 — Acetylcholine System Support

THE CENTURION — The Anticholinergic Component Requires Its Own Recovery Track

PCP's muscarinic acetylcholine antagonism is an aspect of PCP recovery that no other guide in this series addresses — because no other substance has this combination. The sustained anticholinergic load of chronic PCP use produces cognitive effects (impaired memory, impaired attention) and the dry/anhidrotic peripheral effects through the acetylcholine system. Recovery requires supporting this system back to baseline:

- Alpha-GPC (300–600mg morning): the most bioavailable acetylcholine precursor. Directly provides the choline needed for acetylcholine synthesis. Particularly important for memory and cognitive recovery from PCP's anticholinergic burden. The compendium identifies Alpha-GPC as a primary supplement in the cognitive recovery stack.
- CDP-Choline (250mg, morning — already in the Brain Stack above): dual function — dopamine D2 upregulation AND acetylcholine precursor. The combination with Alpha-GPC provides comprehensive cholinergic support.
- Huperzine-A (50–100mcg, with physician guidance): acetylcholinesterase inhibitor — prevents the breakdown of available acetylcholine. Should not be combined at high doses with Alpha-GPC and CDP-Choline as the combined cholinergic effect may be excessive. Use one high-dose approach OR low-dose combinations.
- Lion's Mane mushroom (500–1000mg daily): BDNF and NGF stimulation — the compendium names it specifically for NGF support. NGF (Nerve Growth Factor) is required for cholinergic neuron health. Basal forebrain cholinergic neurons are particularly affected by the anticholinergic load of sustained PCP use.

4.5 — Neuroprotective and Antioxidant Stack

PCP's combined dopaminergic, NMDA, and anticholinergic mechanisms produce substantial oxidative stress in the brain. The neuroprotective stack addresses this:

- NAC (already above — serving double duty): glutathione precursor providing brain antioxidant support.
- Alpha-lipoic acid (300mg morning, away from food): blood-brain barrier crossing mitochondrial antioxidant. Relevant for the metabolic stress of PCP's complex receptor disruption.
- EGCG (green tea extract, 400–800mg morning): same NMDA-related neuroprotective evidence as in MDMA recovery — antioxidant protection for neurons during receptor system recovery.
- Semax peptide (intranasal, Week 2 onwards): the compendium's Semax entry: BDNF and NGF upregulation; cognitive enhancement; prefrontal cortex support. For PCP recovery — where prefrontal BDNF suppression from prolonged NMDA antagonism may be the most significant persistent cognitive impairment — Semax is the most targeted available intervention.

4.6 — Sleep Architecture

The insomnia of PCP cessation has multiple simultaneous drivers: NMDA rebound (glutamate hyperexcitability disrupting sleep), dopaminergic stimulant crash (the same disrupted sleep as cocaine/meth cessation), anticholinergic rebound (cholinergic overactivity that contributes to REM disruption), and ongoing psychosis if present.

- Quetiapine (25–50mg prescription, physician guidance): addresses sleep, anxiety, and the psychosis dimension simultaneously. The most useful pharmacological sleep tool for acute PCP cessation where psychosis is also present.
- Magnesium glycinate 400mg + glycine 3g + melatonin 0.5mg: the standard sleep stack from the compendium; non-pharmacological support once quetiapine is being tapered.
- L-Theanine 200mg evening: reduces the NMDA-rebound cognitive hyperactivity that prevents sleep onset.
- Sonic protocol: delta binaural beats (0.5–4 Hz); 60–70 BPM maximum; warm acoustic textures. The dissociative withdrawal sonic architecture from the compendium. No sudden dynamics. No music associated with PCP use.

4.7 — Peptide Support

Peptide	Why it matters for PCP recovery	Timing	Notes
BPC-157	Dopamine AND serotonin system modulation (addressing both the dopaminergic stimulant and the NMDA recovery dimensions); NMDA receptor pathway modulation; gut-brain axis repair; neuroprotection across multiple mechanisms	Day 1 onwards — daily	BPC-157's multi-system coverage addresses PCP's multi-receptor damage simultaneously. Its documented dopamine system modulating properties address the stimulant component; its NMDA-adjacent effects support the dissociative recovery track. The most appropriate single peptide for PCP's complex pharmacology. Oral.
Semax	BDNF and NGF upregulation — the primary intervention for PCP's prefrontal BDNF suppression and the cholinergic neuron NGF-dependence that the anticholinergic load has stressed	Week 2 onwards	For heavy PCP use histories with documented cognitive impairment — impaired memory, slow processing, executive function deficits — Semax is the most targeted peptide available. The prefrontal cortex repair that the cognitive consequences of prolonged PCP-NMDA antagonism require is BDNF and NGF dependent. Intranasal.
Selank	GABA and serotonin modulation; non-	Weeks 1–8 as needed	The dual anxiety drivers of PCP cessation (NMDA

	addictive anxiolytic; the anxiety of NMDA rebound and the stimulant crash converge to produce severe anxiety in early PCP recovery — Selank addresses this without addiction risk		rebound AND dopaminergic crash) make the early recovery anxiety more severe and more persistent than in pure dissociative or pure stimulant recovery. Selank provides the non-addictive anxiolytic support for this convergent anxiety. Intranasal.

Section 5 — The PCP Craving — Understanding and Interrupting

THE CENTURION — The Dual Craving: Dissociative Escape AND Stimulant Intensity — Both Simultaneously

PCP cravings are the most complex in this guide series because they are the product of two simultaneous craving architectures that most frameworks address separately. The person craving PCP is simultaneously craving the dissociative escape (the NMDA mechanism that quiets the ordinary self) AND the stimulant intensity (the dopaminergic arousal that makes the ordinary baseline feel insufficient). Both must be addressed in the interrupt.

Trigger	The underlying state	The interrupt
Wet / sherm environmental cues (smell of the cigarette, specific contexts where it was smoked)	The most potent and fastest-firing PCP craving triggers. The phantom smell phenomenon the compendium describes for ketamine applies with greater intensity to PCP — the specific chemical-tinged smell of a PCP cigarette is one of the most strongly conditioned olfactory cues in this guide series.	Environmental redesign that addresses olfactory cues specifically: removing all cigarette papers, tobacco, and any materials associated with wet preparation from the home. The geographic cues require the same attention as crack cocaine and fentanyl — specific locations where wet was obtained or smoked fire the craving through environmental conditioning.
The flatness of ordinary consciousness — the dual crash	The convergence of NMDA rebound (which makes ordinary cognition feel hyperactive and anxious) with dopaminergic crash (which makes ordinary reward feel insufficient) produces a state where the sober world is simultaneously too loud AND too dull. This is the specific phenomenological experience of early PCP cessation that no other guide in the series addresses.	The dual natural architecture: IMMEDIATE intense physical exercise for the dopaminergic crash (the Warrior needs a real-intensity signal), FOLLOWED by the 4-7-8 breath and magnesium for the NMDA rebound anxiety. The dual craving requires a sequenced response — intensity first, calm second — rather than either alone.
Pain — physical or emotional	PCP's NMDA analgesia provided complete pain relief for many users. When pain is present — physical chronic pain or the emotional pain of difficult circumstances — the craving fires the pharmacologically trained response: PCP was the	Pain management plan (same as kratom and fentanyl guides): non-opioid pain management, physical therapy, exercise-generated endorphin analgesia, magnesium for the NMDA component of pain signaling. The craving is pointing at real

	agent that made this bearable.	pain — address the pain, not just the craving.
Psychosis residue — when perceptual disturbances fire craving	An unusual feature of PCP recovery: residual perceptual phenomena (HPPD-like visual disturbances, cognitive disorganization, or mood dysregulation from lingering PCP effects) can paradoxically drive craving for more PCP. The person experiencing disturbing PCP residue may seek PCP to 'smooth out' the disturbance — the same drug that caused it.	This is the most counterintuitive PCP craving dynamic. Name it: 'What I am experiencing is PCP residue from prior use, not a reason to use more PCP. More PCP will worsen and extend the residue. Sleep and time are the resolution.' Antipsychotic medication, if prescribed for residual psychosis, directly addresses this craving's source.

THE PCP CRAVING INTERRUPT — 5-MINUTE PROTOCOL

1. NAME WHICH CRAVING IS FIRING. Dissociative escape (need for the quiet, the disconnection, the pain relief)? Stimulant intensity (need for the arousal, the power, the loudness)? Both simultaneously? The name determines the parallel response components.
2. HIGH-INTENSITY PHYSICAL INTERRUPT FIRST. Sprints, push-ups, cold water. This addresses the dopaminergic stimulant dimension — the Warrior needs a real signal before anything else can be effective. Do not skip this step.
3. 4-7-8 BREATHING AFTER THE PHYSICAL — SIX CYCLES. The NMDA rebound anxiety that co-occurs with the stimulant crash requires vagal brake activation AFTER the physical intensity step has provided the dopaminergic signal.
4. TAKE NAC, MAGNESIUM, AND TYROSINE. Addressing all three dimensions: NAC for NMDA glutamate homeostasis, magnesium for NMDA channel modulation, tyrosine for the dopamine precursor supply.
5. CONTACT. PCP use is often social and geographically specific. The person, the place, and the substance form a conditioned unit. Contact with someone outside that unit provides social interruption of the conditioned context.
6. FOR PAIN-DRIVEN CRAVING: Execute the pain management plan. The complete analgesia of PCP was real. The recovery must provide a real alternative.
7. FOR PSYCHOSIS-RESIDUE CRAVING: Take your prescribed antipsychotic if applicable. Name the phenomenon clinically: this is PCP residue in my fat tissue being metabolized. It will diminish. More PCP will extend it.

Section 6 — Porting the Addiction: Building the Dual Natural Architecture

THE CENTURION — Two Architectures Must Be Built Simultaneously — Because PCP Was Doing Two Things

The compendium's C-28 substance masking analysis produces a dual signal for PCP: the dissociative escape (same as ketamine — DMN suppression, pain relief, the quieting of ordinary self-referential consciousness) AND the stimulant intensity (same as cocaine and meth — the Warrior's need for genuine arousal, the flooding of the reward circuit beyond its ordinary reach). Both must be addressed. Neither alone is sufficient.

Track 1: The Dissociative Escape Architecture

From the ketamine guide — applied identically here because the NMDA mechanism is the same:

- Meditation (absorption states): the most direct natural access to DMN suppression. Daily practice. The ketamine guide's contemplative tradition framework applies. The compendium: 'the progressive states of meditative concentration access flow through the deliberate application of sustained attention to a worthy object.'
- NSDR / yoga nidra: the hypnagogic state between waking and sleep. 20–30 minutes daily. The closest non-pharmacological equivalent to the quieting of consciousness that NMDA antagonism produced.
- Holotropic breathwork: produces genuine altered states through CO2/O2 balance. Non-chemical access to the territory PCP was opening through receptor blockade. With trained guide.
- Nature immersion and awe: the DMN suppression that genuinely large-scale natural environments produce — mountains, ocean, open sky. The ketamine guide's 'awe practice' applies directly.
- Pain management (if pain relief was primary): exercise-generated endorphin analgesia; physical therapy; magnesium's NMDA pain modulation; low-dose naltrexone for pain conditions.

Track 2: The Stimulant Intensity Architecture

From the cocaine, meth, and crack cocaine guides — applied to the dopaminergic component of PCP:

- High-intensity competitive exercise: vigorous training with genuine stakes. The Warrior's dopamine requirement is real and requires real-intensity

response. The natural dopamine generated through genuine physical challenge addresses the DAT-inhibition component's recovery.

- Competition in any domain: the social-stakes dopamine of genuine competition — sport, business, creative, martial arts. Genuine risk, genuine reward.
- Mastery at the skill frontier: sustained engagement at the challenge-skill edge. Flow state dopamine through creative or technical mastery.
- Service: the sustained meaning and dopamine-oxytocin combination of genuine contribution. For the PCP recovery population — often concentrated in communities affected by violence, poverty, and the same structural conditions that made PCP available — peer recovery support and community service are particularly meaningful applications.

Track 3: The Acetylcholine Restoration Architecture

THE CENTURION — The Third Track That No Other Guide Requires — Because No Other Substance Has the Anticholinergic Dimension

The acetylcholine system that PCP was antagonizing is the primary system for memory formation, executive attention, and the specific quality of cognitive clarity that the anticholinergic load of PCP was impairing. Building access to acetylcholine-mediated cognitive states through natural means:

- Deliberate memory practice: the explicit engagement of episodic memory — journaling, storytelling, remembering specific events in detail — activates the cholinergic circuits in the hippocampus and prefrontal cortex that PCP's muscarinic antagonism was suppressing.
- Sustained attention practices: reading long-form material that requires sustained attention (not social media); chess; any practice requiring extended focus without breaks. Cholinergic circuits are the substrate of sustained attention. Practice rebuilds the system.
- Dietary choline sources: eggs, liver, fish, legumes — the dietary precursors to acetylcholine production. The recovery diet should include choline-rich foods alongside the Alpha-GPC and CDP-Choline supplements.
- Physical coordination and skill learning: new motor skills require intensive cholinergic circuit engagement. Martial arts, dance, musical instrument learning — any motor skill with genuine complexity builds the acetylcholine system through use.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	PCP-specific notes
Acute Medical Management (Days 0–7)	Section 3 of this guide; physician; psychiatrist if psychosis	Prolonged psychosis management; sleep; cardiac monitoring; CYP2B6 testing	Psychosis management takes precedence over all other recovery work in this window. Antipsychotic if indicated. Sleep is the highest priority non-pharmacological intervention — quetiapine serves both. CYP2B6 testing from Day 1 if possible: the metabolizer status determines the expected psychosis duration and informs medication selection.
Biological Foundation (Days 1–14)	Protocol 07, Protocol 09, Protocol 17	Triple stack: NMDA (NAC + Mg), dopamine (tyrosine + Brain Stack), acetylcholine (Alpha-GPC); 5-MTHF; sleep stack	Three parallel supplement tracks running simultaneously from Day 1 — the NMDA recovery track, the dopaminergic stimulant track, and the acetylcholine support track. This is more complex than any other guide in the series because PCP requires all three. Prioritize: magnesium and NAC from Day 1 (NMDA). Alpha-GPC and tyrosine from Day 2. Brain Stack from Week 1.
Genomic Mapping (Weeks 1–4)	CYP2B6, CYP3A4; COMT; DRD2; MTHFR; BDNF	Dissociative Logjam (CYP2B6); psychosis risk (COMT + DRD2); dopaminergic constitution; synthesis support	CYP2B6 is the highest priority test in PCP recovery — it explains the specific psychosis duration and severity and informs all future medication decisions involving CYP2B6-metabolized drugs. COMT + DRD2 determines the primary natural architecture direction (stimulant vs. dissociative emphasis).
Dual Natural Architecture (Months 1–6)	Section 6 of this guide; ketamine guide Section 6; cocaine guide Section 6	Build both dissociative escape architecture AND stimulant intensity architecture simultaneously	No other guide requires building two natural architectures simultaneously. The sequence: begin with the physical intensity architecture (stimulant track — easier to access quickly, more immediately gratifying), then build the contemplative architecture (dissociative track — slower to develop, more sustainable for the

			escape dimension).
Cognitive Recovery (Months 3–12)	Semax; Alpha-GPC; lion's mane; deliberate cognitive practice; ADHD evaluation	Prefrontal cortex repair; memory system recovery; acetylcholine circuit rebuild; ADHD treatment if indicated	PCP's prefrontal and cholinergic consequences may require the longest explicit recovery work of any substance in this series — 6–12 months for significant cognitive improvement in heavy use histories. Semax + exercise as the primary BDNF interventions. Explicit cognitive practices (memory, sustained attention) rebuild the acetylcholine system through use.
Community and Long Game (Months 6+)	Protocol 20, Protocol 21, C-28; peer recovery support	The Warrior's intensity fully engaged; the dissociative territory accessible through earned practice; the cognitive recovery complete; the community rebuilt	PCP recovery is long-game work precisely because of the cognitive dimension. The person at 12 months with rebuilt prefrontal function, both natural architectures operational, and the cholinergic memory system recovering — has access to a clarity of consciousness that PCP was approximating through receptor blockade. The real thing is clearer.

Section 8 — The Medical and Community Context

PCP occupies a specific historical and community context that clinical medicine has largely failed to address adequately — mirroring, in some respects, the failures the compendium documents with crack cocaine.

8.1 — The Community Dimension

THE CENTURION — The Compendium's Crack Cocaine Framework Applied Here: Punishment Substituted for Medicine

PCP's epidemic periods — the late 1970s and 1980s, with regional resurgences — disproportionately affected urban, lower-income, predominantly Black communities, particularly in Los Angeles, Washington DC, Philadelphia, and Detroit. The treatment system's response to PCP misuse in these communities has historically prioritized law enforcement over medicine.

The specific failures: treatment specifically for PCP use disorder is rare even now. Most substance use disorder programs do not have PCP-specific protocols. The CYP2B6 dissociative logjam that determines why some individuals experience prolonged, severe PCP psychosis is rarely tested in treatment populations who need it most. The cognitive consequences of heavy PCP use — the prefrontal and cholinergic impairment that affects employment, relationships, and the capacity to engage with recovery programs — are rarely addressed clinically.

The Recovery Kernel's position, stated plainly: the person who developed PCP use disorder in conditions of poverty, community disruption, and inadequate treatment infrastructure was not failed by their own constitution. They were failed by systems that chose enforcement over medicine and that, when medicine was offered, rarely offered the medicine that the specific pharmacology of PCP required.

8.2 — Co-Occurring Conditions Common in PCP Recovery

- PTSD: trauma is extremely common in the communities most affected by PCP and in the experiences that PCP use disorder develops within. The dissociative and analgesic properties of PCP specifically buffer trauma symptoms. PTSD treatment is a concurrent recovery track.
- ADHD: the DAT-mediated stimulant component of PCP may have been self-medicating genuinely untreated ADHD. Evaluation and non-stimulant treatment (atomoxetine, guanfacine) are appropriate where ADHD is confirmed.
- Tobacco / nicotine: PCP is predominantly smoked in the wet/sherm form, usually on tobacco or cannabis. The nicotine dependence that co-occurs with

PCP smoking requires its own treatment — varenicline, NRT, or bupropion (note CYP2B6 metabolizes bupropion — the same gene as PCP — metabolizer status affects bupropion dosing significantly).

- Cannabis use disorder: many people who use wet/sherm use cannabis as the base rather than tobacco. Cannabis co-occurring with PCP use disorder requires evaluation.

8.3 — Clinical Resources

- SAMHSA Treatment Locator (findtreatment.gov or 1-800-662-4357): for general substance use disorder treatment. PCP-specific programs are rare but some urban addiction medicine programs have specific experience.
- Psychiatric emergency: if prolonged psychosis — crisis evaluation at any emergency department. Specifically name PCP as the substance.
- CYP2B6 testing: through any clinical laboratory or pharmacogenomic testing service. This is the single most actionable genetic test in PCP recovery and one that most treating physicians will not think to order.
- Crisis: 988 (Suicide and Crisis Lifeline) | 911 for medical emergencies | SAMHSA: 1-800-662-4357

A Final Word

THE CENTURION — The Clarity Was Real. The Instrument Was Borrowed. Recovery Is Owning It.

PCP is the hardest substance in this guide series to write a closing for, because it does not fit any single story. It is simultaneously the substance that provided the most pharmacologically complete escape available — NMDA analgesia that blocked every kind of pain simultaneously — and the substance that produced the most publicly visible and most misunderstood behavioral profile of any substance of misuse. The person who used it was not the person the public saw.

The compendium's C-37 framework asks: what was the substance providing? For PCP, the answer is complex and honest: it provided the silence of NMDA receptor blockade, the intensity of dopaminergic flooding, the perceptual distinctiveness of sigma-1 activation, and the anticholinergic blunting of embodied stress. All four, simultaneously. Nothing else in this guide series does all four at once.

That combination was seeking something real. The silence from unbearable noise. The intensity that the baseline never provided. The pain relief that the body never generated adequately on its own. The perceptual distinctiveness that ordinary consciousness denied.

Recovery is not denying that these needs were real. Recovery is building direct access to what was being sought through means that do not produce prolonged psychosis, do not accumulate in fat tissue and re-emerge weeks later, and do not require the person to become invisible to a healthcare system that never developed a treatment for what they were experiencing.

The meditative silence is real — and accessible through practice, without NMDA blockade. The intensity is real — and available through genuine challenge and competition, without DAT inhibition. The pain relief is real — and achievable through endorphin architecture and genuine pain management, without the analgesia that allowed injuries to go unnoticed. The perceptual distinctiveness is real — and accessible through awe, creative flow, and contemplative depth, without sigma-1 agonism.

The compendium names CYP2B6 as the Dissociative Logjam gene. In PCP recovery, the logjam is not only pharmacological. It is the gap between what the person needed and what was available. Recovery builds the path around the logjam.

The clarity was always real. The instrument was borrowed. Recovery is owning what was borrowed — directly, permanently, without interest paid in psychosis.

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Refer to the Recovery Kernel Complete Centurion Compendium and the Ketamine Guide for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357